REDISCOVERING URINE ELECTROLYTES FOR DIFFERENTIAL DIAGNOSIS AND PROGNOSIS OF AKI

ETIENNE MACEDO
UNIVERSITY OF SÃO PAULO
Factors affecting the sensitivity and specificity of urine electrolytes to determine

- reversibility of acute kidney injury
- severity

Discuss the interpretation of urine electrolytes in acid-base imbalances.

Propose the use of SIDu to monitor tubular acidifying capacity in AKI.
Urine electrolytes

- Differential diagnosis of natremia disorders
- Correct interpretation of urinary electrolytes
- Pre-renal versus acute tubular necrosis
Diagnostic Value of Urinary Sodium, Chloride, Urea, and Flow

Robert W. Schrier

B  ACUTE DETERIORATION IN RENAL FUNCTION

- Prerenal azotemia
  - Renal hypoperfusion
- Postrenal azotemia
  - Urinary tract obstruction
- Glomerular, vascular, interstitial
  - Hematuria, RBC casts, proteinuria, pyuria
- Acute tubular necrosis
  - Ischemia and/or toxic insults
Urinary sodium reflects ECFV

- modest changes in ECFV (extra-cellular fluid volume) or total body sodium
  - stimulation of renin-angiotensin-aldosterone system - sympathetic nervous system
    - Decreases urinary excretion of Na
      - Urine Na concentration
      - FE Na
When Urinary Sodium does not Reflect ECFV (or Total Body Sodium)

- Diuretics
- Bicarbonaturia — in metabolic alkalosis or proximal tubular acidosis
- Increase in solute excretion may also increase urinary sodium losses by the normal kidney
  - Glucosuria
  - Mannitol
Chronic Kidney Disease

- GFR < 60 ml/min
  - the renal response not maximal
  - Can take days
  - can still decrease in patients with CKD who are not at end-stage
Established Acute Tubular Necrosis

- Urinary sodium concentration will not be minimal, even with substantial ECFV depletion
Causes of falsely low Urine Na in patients with an intrinsic cause of AKI

- Selected causes of acute tubular necrosis
  - Early in contrast-mediated acute renal dysfunction
  - Rhabdomyolysis
  - Myoglobinuria, hemoglobinuria
  - Nonoliguric acute tubular necrosis
- Acute glomerulonephritis
- Acute interstitial nephritis
- Early in sepsis — functional AKI?
Low Urine Na with high ECFV
Underfilling

- Stimulus of the normal kidney to retain sodium is not ECFV depletion or even decreased total plasma volume
  - Arterial baroreceptors in the carotid sinus, aortic arch, and juxtaglomerular apparatus are unloaded with reversal of tonic inhibition to central nervous system
  - Can be associated with ECFV expansion
    - Decrease in stroke volume
    - Primary systemic arterial vasodilation

- Differentiate a reversible renal dysfunction (“pre-renal”) with acute tubular necrosis (ATN)
  - Cannot by a parameter to guide fluid resuscitation
Diagnostic Value of Urinary Sodium, Chloride, Urea, and Flow

Robert W. Schrier

A CAUSES OF PRERENAL AZOTEMIA

ECF volume
- Renal losses
- Third space losses
- Gastro-intestinal losses

ECF volume with arterial underfilling
- Cardiac output
  - Myocardial infraction
  - Pericardial tamponade, constrictive pericarditis
- Systemic arterial vasodilation
  - Cirrhosis
  - Sepsis
Urinary diagnostic indices in AKI

$FE_{Na}$ vs $FE_{UREA}$

- The FENa < 1.0 in 85 to 94% of patients with prerenal azotemia
  - within 24 to 72 h
  - reversal of kidney function secondary to interventions:
    - such as fluid resuscitation or
    - improved cardiac output.

- Did not reverse their sCr and thus had oliguric ATN
  - FENa < 1.0 in only 0 to 4%.

What about the FE Urea?
Fractional Excretion of Urea

- More helpful than FENa in distinguishing prerenal azotemia from ATN in patients on diuretics
- Urea reabsorption in prerenal states
  - ECFV depletion
  - Heart failure
  - Cirrhosis
  - Is enhanced in the proximal tubule before the sites of diuretic action in the downstream tubule

FEUN vs FENA in 102 episodes of ARF

three groups:

- Prerenal no diuretics n= 50
  - 92% FENA < 1%
- Prerenal with diuretics n= 27
  - 48% FENA < 1%
- ATN n = 25
Significance of The Fractional Excretion of Urea in The Differential Diagnosis of Acute Renal Failure
CHRISTOS P. CARVOUNIS, SABEEHA NISAR, and SAMERAH GURO-RAZUMAN

- Prerenal no diuretics n= 50
  - 92% FENa < 1%

- Prerenal with diuretics n= 27
  - 48% FENa < 1%
  - 89% Feurea < 85%

- ATN n = 25

- FE Urea identical in the two pre-renal groups
  - (27.9 2.4% vs. 24.5 2.3%)
  - ATN (58.6%)
Prospective study

- Feur vs FENa - transient and persistent AKI
- 99 patients AKI
  - (>=30% sCr within 1 week)
  - returned to baseline within 7 days
Performance of FE <= 35% and FENA <= 1% for the Diagnosis of Transient Acute Kidney Injury

Irrespective of diuretic intake:

FENa was less in T-AKI than P-AKI (2±4% vs 5±6%; P=0.001)

FEur was similar in T-AKI (29±19%) and P-AKI (32±19%; P=0.3).

<table>
<thead>
<tr>
<th></th>
<th>No Diuretics</th>
<th>Diuretics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity (%)</td>
<td>48</td>
<td>79</td>
</tr>
<tr>
<td>Specificity (%)</td>
<td>75</td>
<td>33</td>
</tr>
<tr>
<td>Positive predictive value (%)</td>
<td>79</td>
<td>71</td>
</tr>
<tr>
<td>Negative predictive value (%)</td>
<td>43</td>
<td>44</td>
</tr>
</tbody>
</table>

ROC Curves For FEur Fena for Diagnosis of Transient AKI Patients With and Without Diuretic Intake.

No diuretic – 0.56 vs 0.86
Diuretic – 0.57 vs 0.75

“Prerenal azotemia is classically defined as decreased GFR resulting from renal hypoperfusion in a structurally intact kidney, which is rapidly reversible when the underlying cause is corrected”

✓ No consensus definition for PRA

✓ “reversible increase in serum creatinine and urea concentrations,

✓ characterized by intact renal parenchymal function but renal hypoperfusion”
Transient Azotemia

Persistent Azotemia

Pre-renal Azotemia

Acute tubular necrosis (ATN)

Reversible:
Functional

Intact Parenchyma
Histopathological
How Studies Classify Prerenal Patients

“Prerenal AKI was defined as an abrupt decline in baseline kidney function that improved to 10% of baseline after fluid resuscitation and/or hemodynamic manipulation within 48 h.”
## Definitions of PRA used in studies differentiating PRA and ATN

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Test</th>
<th>Definitions of PRA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perlmutter</td>
<td>1959</td>
<td>Urine-serum urea nitrogen</td>
<td>Oliguria and azotemia lasting less than 48 hrs.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ratio</td>
<td></td>
</tr>
<tr>
<td>Espinel</td>
<td>1976</td>
<td>FE-Na</td>
<td>Prompt increase in urinary output and creatinine clearance effected by hemodynamic</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>improvement.</td>
</tr>
<tr>
<td>Miller</td>
<td>1978</td>
<td>Urinary indices</td>
<td>Return of renal function to normal within 24 to 72 hrs after correction of</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>hemodynamics.</td>
</tr>
<tr>
<td>Platt</td>
<td>1991</td>
<td>Doppler ultrasound</td>
<td>Clinical judgment (definitions not mentioned).</td>
</tr>
<tr>
<td>Chew</td>
<td>1993</td>
<td>Urinary enzymes</td>
<td>Rapid recovery of renal function after treatment of hypotension or dehydration.</td>
</tr>
<tr>
<td>Steinhauslin</td>
<td>1994</td>
<td>FE-lithium, FE-UA</td>
<td>Decrease in plasma creatinine toward normal values within 72 hrs of correction of</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>hemodynamic abnormalities.</td>
</tr>
<tr>
<td>Izumi</td>
<td>2000</td>
<td>Doppler ultrasound</td>
<td>Not clearly mentioned, but FENa used.</td>
</tr>
<tr>
<td>Carvounis</td>
<td>2002</td>
<td>FE-urea</td>
<td>Prompt increase in urinary output and creatinine clearance after hemodynamic</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>improvement.</td>
</tr>
<tr>
<td>Parikh</td>
<td>2004</td>
<td>Urinary IL-18</td>
<td>Multiple definitions but included improvement after treatment.</td>
</tr>
<tr>
<td>Pepin</td>
<td>2007</td>
<td>FE-Na, FE-urea</td>
<td>Two of 4 criteria (history, physical findings, urine analysis, rapid return to</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>baseline renal function within 7 days).</td>
</tr>
<tr>
<td>Perazella</td>
<td>2008</td>
<td>Urine microscopy</td>
<td>Improvement to baseline after fluid resuscitation and/or hemodynamic manipulation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>within 48 hrs.</td>
</tr>
<tr>
<td>Nickolas</td>
<td>2008</td>
<td>Urinary NGAL</td>
<td>Resolved within 3 days or FENa &lt;1%</td>
</tr>
</tbody>
</table>
Is it important to diagnose transient AKI?
Transient vs Prolonged AKI

Difference in Outcomes

Rapid Reversal of AKI and Hospital Outcomes: A Retrospective Cohort Study

- Tian, J Am J Kidney Dis 53: 974-981; 2009

Transiente Azotemia is Associated with a high risk of death in hospitalized patients

- Uchino, Nephrol Dial Transplant; 2010
Rapid Reversal of Acute Kidney Injury and Hospital Outcomes: A Retrospective Cohort Study

Jianmin Tian, MD, MPH, Fidel Barrantes, MD, Yaw Amoateng-Adjepong, MD, PhD, and Constantine A. Manthous, MD

Total adult admissions to Medicine: 16,039

Excluded Patients:
- HD or PD: 128
- Admitted to ICU initially: 1,968
- Multiple admissions: 5,406*
- Insufficient # SCr measurements: 2,504

Eligible patients: 6,033

SCr increased ≥ 0.3 mg/dL within 48 h?

Yes

AKI: 735

No

Non-AKI: 5,298

* Multiple admissions: 5,406
Rapid Reversal AKI

443 rapid reversal AKI – more than 0.3mg/dL decrease within 48 h

292 prolonged AKI – no improvement after 48 h of the diagnosis

Tian, J Am J Kidney Dis 2009
Rapid Reversal AKI

Controls:
No AKI no AKI
ate ICU admission or during ICU stay

Admission SCr normal?
Yes
3,901
Non-AKI: 5,298

No
Elevated SCr: 1,397

SCr increased ≥ 0.3 mg/dL, but > 48 h later?
Yes
25
Controls: 3,876

No

SCr decreased ≥ 0.3 mg/dL within 48 h?
Yes
602

No
395

SCr returned to normal within 48 h?
Yes
281

No
321

Tian, J Am J Kidney Dis 2009
### Rapid Reversal AKI

#### Older More comorbidity Worse outcomes Than controls

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Patients With AKI With SCr That Decreased ≥ 0.3 mg/dL Within 48 h</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No. of patients</strong></td>
<td>3,876</td>
<td>443</td>
</tr>
<tr>
<td><strong>Age (y)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥65</td>
<td>1,842 (47.5)</td>
<td>304 (68.6)</td>
</tr>
<tr>
<td>&lt;65</td>
<td>2,034 (52.5)</td>
<td>139 (31.4)</td>
</tr>
<tr>
<td>Mean ± SE</td>
<td>62 ± 0.3</td>
<td>71 ± 0.7</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>1,662 (42.9)</td>
<td>221 (49.9)</td>
</tr>
<tr>
<td>Women</td>
<td>2,214 (57.1)</td>
<td>222 (50.1)</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>2,575 (66.4)</td>
<td>302 (68.2)</td>
</tr>
<tr>
<td>African American</td>
<td>621 (16.0)</td>
<td>95 (21.4)</td>
</tr>
<tr>
<td>Other</td>
<td>620 (17.6)</td>
<td>44 (10.4)</td>
</tr>
<tr>
<td><strong>Deyo-Charlson comorbidity index score‡</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>2,134 (55.1)</td>
<td>100 (22.6)</td>
</tr>
<tr>
<td>1-2</td>
<td>1,437 (37.1)</td>
<td>222 (50.1)</td>
</tr>
<tr>
<td>3-4</td>
<td>197 (5.1)</td>
<td>81 (18.3)</td>
</tr>
<tr>
<td>≥5</td>
<td>103 (2.7)</td>
<td>48 (10.9)</td>
</tr>
<tr>
<td><strong>Transfer to intensive care unit</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>184 (4.7)</td>
<td>160 (36.1)</td>
</tr>
<tr>
<td><strong>Mean length of stay (d)</strong></td>
<td>5 ± 0.1</td>
<td>14 ± 0.6</td>
</tr>
<tr>
<td><strong>Hospital mortality</strong></td>
<td>49 (1.3)</td>
<td>59 (13.3)</td>
</tr>
<tr>
<td><strong>Discharge§</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Home</td>
<td>3,049 (78.7)</td>
<td>202 (45.6)</td>
</tr>
<tr>
<td>Extended-care facility</td>
<td>655 (16.9)</td>
<td>170 (38.4)</td>
</tr>
<tr>
<td>Hospice care</td>
<td>16 (0.4)</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

*Tian, J Am J Kidney Dis 2009*
Rapid Reversal AKI
Associated mortality

<table>
<thead>
<tr>
<th>AKI Type</th>
<th>Unadjusted Odds Ratio (95% confidence interval)</th>
<th>Adjusted Odds Ratio (95% confidence interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AKI fully reversed*†</td>
<td>12.9 (7.9-21.1)</td>
<td>4.4 (2.6-7.3)</td>
</tr>
<tr>
<td>AKI with SCr that did not return to normal*</td>
<td>11.3 (7.0-18.0)</td>
<td>4.4 (2.7-7.1)</td>
</tr>
<tr>
<td>AKI with SCr that did not decrease ≥ 0.3 mg/dL within 48 h*</td>
<td>16.1 (10.7-24.4)</td>
<td>8.0 (5.4-11.8)</td>
</tr>
<tr>
<td>Age (≥65 y)</td>
<td>4.3 (2.9-6.4)</td>
<td>3.2 (2.1-4.8)</td>
</tr>
<tr>
<td>Intensive care unit transfer</td>
<td>7.5 (5.5-10.3)</td>
<td>4.0 (2.8-5.8)</td>
</tr>
<tr>
<td>Deyo-Charlson comorbidity index score‡</td>
<td>2.5 (1.8-3.5)</td>
<td>1.4 (1.1-1.6)</td>
</tr>
</tbody>
</table>

Tian, J Am J Kidney Dis 2009
Transient azotaemia is associated with a high risk of death in hospitalized patients

Shigehiko Uchino¹, Rinaldo Bellomo², Sean M. Bagshaw³ and Donna Goldsmith²
Transient Azotemia

Risk for hospital mortality 3 times higher for patients with prolonged AKI.

Table 4. Multivariate logistic regression analysis for hospital mortality

<table>
<thead>
<tr>
<th>Variables</th>
<th>Odds ratios (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>1.036 (1.031–1.041)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Male gender</td>
<td>1.199 (1.060–1.356)</td>
<td>0.0038</td>
</tr>
<tr>
<td>Readmission</td>
<td>1.860 (1.636–2.115)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Emergency admission</td>
<td>1.543 (1.327–1.795)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>ICU admission</td>
<td>3.181 (2.500–4.048)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>5.007 (3.826–6.552)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Baseline creatinine, mg/dL</td>
<td>1.514 (1.332–1.722)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Operation</td>
<td>0.809 (0.665–0.983)</td>
<td>0.033</td>
</tr>
<tr>
<td>Renal condition</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No AKI</td>
<td>1.000 (Reference)</td>
<td></td>
</tr>
<tr>
<td>ATN</td>
<td>6.070 (5.305–6.944)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>TA</td>
<td>2.264 (1.856–2.762)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Uchino, Nephrol Dial Transplant (2010)
Transient Azotemia

Days with AKI and risk for mortality

Uchino, Nephrol Dial Transplant (2010)
Transient AKI

- Is common (4-6% of hospitalized patients).
- Independent association with increased mortality.
- Associated with higher hospital mortality compared to patients with no AKI.
- Even one day of AKI had a significantly increased odds ratio for hospital mortality.
Functional vs Histopathological concepts

Reversible:
- Transient Azotemia

Intact Parenchyma
- Pre-renal Azotemia

Histopathological:
- Persistent Azotemia
- Acute tubular necrosis (ATN)
Sensitivity and Specificity of a Single ED Measurement of Urinary NGAL for Diagnosing AKI

Prerenal azotemia:
new-onset increase in sCr level (RIFLE)
And
Resolved within 3 days with treatment aimed at restoring perfusion

Urinary NGAL distinguishes pre-renal from intrinsic renal failure and predicts outcomes

Eugenia Singer, Antje Elger, Saban Elitok2, Ralph Kettritz1, Thomas L. Nickolas, Jonathan Barasch3, Friedrich C. Luft1,2 and Kai M. Schmidt-Ott1,

All patients

(n = 145)

Prerenal AKI

(n = 32)

Composite outcome

(n = 2)

No aspect of composite outcome

(n = 30)

Unclassifiable

(n = 38)

Composite outcome

(n = 9)

No aspect of composite outcome

(n = 29)

Intrinsic AKI

(n = 75)

Composite outcome

(n = 38)

No aspect of composite outcome

(n = 37)
NGAL levels were significantly higher in patients with a clinical diagnosis of intrinsic AKI when compared with prerenal AKI.
Biomarker levels in differential diagnosis of AKI and prediction of outcomes.

Median NGAL levels on inclusion and 2 days after inclusion were significantly higher in patients, who later experienced the composite clinical outcome, when compared with all others.
Biomarkers in Predicting Intrinsic AKI vs Prerenal AKI

- NGAL levels effectively discriminated between intrinsic and prerenal AKI
  - area under the receiver-operating characteristic curve 0.87

- An NGAL level
  - over 104 lg/l indicated intrinsic
  - < 47 lg/l – unlikely intrinsic AKI
Current Concepts of Reversibility

- Reversibility with manipulation
  - Fluid
  - Hemodynamics

- Common in certain settings
  - Dehydration
  - Hypotension

- Biomarkers
  - Urine output
  - Changes in BUN/Creatinine/Electrolytes in serum and urine
# Clinical review: Reunification of acid–base physiology

John A Kellum

**Figure 1**

<table>
<thead>
<tr>
<th>Descriptive</th>
<th>Semi-quantitative</th>
<th>Quantitative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Henderson-Hasselbalch</td>
<td>Base Excess</td>
<td>Physical Chemical</td>
</tr>
<tr>
<td>pCO₂ “Fixed acids” H⁺</td>
<td>pCO₂ Buffer Base</td>
<td>pCO₂ SID A&lt;sub&gt;TOT&lt;/sub&gt;</td>
</tr>
<tr>
<td>HCO₃⁻ Anion Gap</td>
<td>SBE</td>
<td>SIG</td>
</tr>
</tbody>
</table>

**Affecters**

**Markers & Derived Variables**
Diagnosing metabolic acidosis in the critically ill: bridging the anion gap, Stewart, and base excess

Christina Fidkowski, MD Æ James Helstrom, MD
Diagnosing metabolic acidosis in the critically ill: bridging the anion gap, Stewart, and base excess

Christina Fidkowski, MD Â€ James Helstrom, MD

Kidney instant monitoring: a new analyzer to monitor kidney function

As the kidney has been classically seen as a “slow” organ in the correction of acid-base disturbances, especially as compared to the “fast” lung.
Kidney instant monitoring: a new analyzer to monitor kidney function

CAIRONI1, 2, T. LANGER1, P. TACCONI2, P. BRUZZONE2, S. DE CHIARA2, F. VAGGINELLI2, L. CASPANI2, C. MARENGHI2, L. GATTINONI
Kidney instant monitoring: a new analyzer to monitor kidney function

CAIRONI1, 2, T. LANGER1, P. TACCON2, P. BRUZZONE2, S. DE CHIARA2, F. VAGGINELLI2, L. CASPANI2, C. MARENGHI2, L. GATTINONI
Kidney instant monitoring: a new analyzer to monitor kidney function

CAIRONI1, 2, T. LANGER1, P. TACCON2E2, P. BRUZZONE2, S. DE CHIARA2, F. VAGGINEL2I2, L. CASPANI2, C. MARENGH2I2, L. GATTINONI
Monitoring of Urine Electrolytes in a Clinical ICU

- 4 bedroom Clinical ICU prospectively recorded daily blood and spot urinary electrolytes from patients with urinary catheters admitted to our ICU

- 235 ICU admissions from oct 2009 - dez 2010

- 200 had urinary catheter for at least the first two days after ICU admission

- 169 included in the analysis

- 17 ESRD
- 6 kidney transplant
- 8 RRT started within 2 days of ICU admission
Urine Na

All patients after ICU admission

After AKI diagnosis or after ICU admission for non-AKI
FE Na

All patients after ICU admission

After AKI diagnosis or after ICU admission for non-AKI
Urine Cl

All patients after ICU admission

After AKI diagnosis or after ICU admission for non-AKI

- AKIN
  - n
  - y

Day after ICU admission
- Day after AKI diagnosis or after ICU admission (non-AKI)
All patients after ICU admission

After AKI diagnosis

SID U

Day after ICU admission

Day after AKI diagnosis or after ICU admission (nonAKI)
Potential benefits of assessing urinary electrolytes

Concept of functional and histopathological to distinguish between transient AKI

Fluid administration: patient vs renal responsiveness

Role of urine electrolytes as a useful tool in the interpretation of acid-base imbalances

SIDu as a monitor of tubular acidifying capacity and early inability of urinary acidification in AKI
Thank you

Acknowledgements

USP Clinical Nephrology Research Group:
- Luis Yu
- Emmanuel Burdman
- Regina Abdulkader
- Alexandre Toledo
- Lilian Freitas
- Deane Carneiro

UCSD Clinical Nephrology Research Group:
- PI: Ravindra Mehta
- Joseé Bouchard
- Rolando Claure
- Sharon Soroko
- Sam Kuo
- Alissar Nabali